Oftedal, Sverre: Elective Localization in the Bronchial Musculature of Streptococci from the Sputum of Cases of Bronchial Asthma, Jour. Am. Med. Assn., May 27, 1916, lxvi, 22.

Preston, J. W.: Report of Cases Pointing to Purulent Infection as a Causative Factor in Mucous Colitis, Jour. Am. Med. Assn., November 27, 1915, lxv, 22.

Irons, E. E., and Brown, E. V. L.: The Etiology of Iritis, Jour. Am. Med. Assn., June 10, 1916, lxvi, 24.

Nathan, P. W.: The Neurological Condition Associated with Polyarthritis and Spondylitis, Am. Jour. Med. Sc., November, 1916, cliii, 5.

A REPORT ON THE ELECTROCARDIOGRAPHIC STUDY OF TWO CASES OF NODAL RHYTHM EXHIBITING R-P INTERVALS.

By James D. Heard, M.D.,

PITTSBURGH,

AND

Arthur E. Strauss, S.B., M.D.,

ST. LOUIS.

R. B. MELLON FELLOW IN INTERNAL MEDICINE, UNIVERSITY OF PITTSBURGH.

(From the Department of Medicine, University of Pittsburgh.)

The rarity of clinical cases of nodal rhythm showing an *R–P* interval seems to us to be sufficient reason for this communication.

The term nodal rhythm is applied to a mechanism in which the seat of impulse formation lies in the *A–V* node or conduction tissues. Although in all hearts the node of Tawara has the inherent power of rhythmic stimulus production, this power is usually kept in abeyance because of the greater rhythmicity of the sino-auricular node, the heart responding to that pacemaker which exhibits the highest rate of impulse formation.

Wilson[1] describes two clinical types of nodal rhythm which, in the sense of Lewis, he calls heterogenetic and homogenetic. The former partakes of the character of a paroxysm of tachycardia with sudden onset and abrupt offset, the heart not under the control of the extrinsic cardiac nerves; the homogenetic type is characterized by a comparatively slow rate, gradual onset and offset, with the heart subject to nerve control.

Our first case belonged to type 1, of which type we find but 6 cases in the literature. These are recorded by Lewis,[2] [3] Rihl,[4]

[1] The Production of *A–V* Rhythm in Man after Administration of Atropin, Arch. Int. Med., 1915, xvi, No. 6, 989–1006.

[2] Auricular Fibrillation and its Relationship to Clinical Irregularity of the Heart, Heart, 1910, i, 306–372.

[3] Paroxysmal Tachycardia Accompanied by the Ventricular Form of Venous Pulse, Heart, 1910, ii, No. 2, 127–147.

[4] Ueber atrioventriculäre Tachycardia beim Menschen, Deutsch. med. Wchnschr., 1907, xxxiv, 632–634.

Cohn,[5] Hume,[6] and Falconer and Dean.[7] The patient was referred to us by Dr. De Witt B. Nettleton, to whom we are indebted for the privilege of publishing the following case history:

CASE I.—W. L. C., male, white, aged fifty-seven years, widower, retired manufacturer.

Chief Complaint. Attacks of palpitation with rapid heart action.

Present Illness. For twenty years patient had had attacks of acceleration and palpitation, lately increasing in frequency. The attacks might come at intervals of every few days, or as often as fifty times in one day. For several months during the past summer the patient had had no attacks, but two weeks ago they had returned. Until recently the attacks seemed to bear no relation to exercise or emotion, but of late they occurred more frequently during fatigue. Although the patient usually walked three or four miles a day, if he walked during an attack extreme fatigue resulted. With the onset of acceleration, there developed an extreme degree of palpitation, and, in the more recent attacks, also a sense of precordial oppression not before present. There was no cyanosis nor dyspnea; no noticeable cough nor edema. Attacks might last a few seconds or for hours, the longest was between three and four hours' duration. Rate during the attack was about 120; the rhythm was regular. If he were standing during an attack, occasionally the patient felt faint. Flatus followed the attacks.

The onset was usually spontaneous, but might be brought on by such change in posture as stooping or by a distended stomach. The palpitation began with a sudden acceleration of heart rate—before this moment the heart beat had been slow, below the threshold of consciousness—the acceleration continued for a variable period, then ceased just as suddenly, was followed by a pause, then a heart thump or two, and finally a resumption of the normal rate without palpitation. The patient can frequently check an attack by holding the breath, by continued deep breathing, or by lying down; these measures, however, are not always efficacious. The effect of vagus pressure was never tried. On the morning of the last examination, lying down caused a short paroxysm, and later a short walk of 300 feet brought on another attack. The patient stated that very rarely was the heart rate accelerated without symptoms.

Past History. Patient had had malaria in early manhood and typhoid at thirty; frequent sore throats but no definite tonsillitis. There had been no scarlet fever nor diphtheria. Renal calculus passed at fifty-two; infection of right kidney followed. He had an

[5] A Case of Paroxysmal Tachycardia, Heart, 1910, ii, No. 2, 170–176.

[6] A Polygraphic Study of Four Cases of Diphtheria with a Pathological Examination of Three Cases, Heart, 1913, v, No. 1, 25–44.

[7] Observations on a Case Presenting a long *A-C* Interval, Associated with Short Paroxysms of Tachycardia arising in the Junctional Tissues, Heart, 1912, iv, No. 2, 137–144.

attack of acute appendicitis, with operation at fifty-three. No cardiorespiratory symptoms between attacks of tachycardia. Troublesome tinnitus was frequent.

Habits. Good.

Family History. Negative.

Physical Examination. Well-developed and well-nourished middle-aged man. Except for the cardiorespiratory-vascular system the examination was practically negative. The heart was not enlarged to percussion. Apex was felt in V interspace about 0.5 cm. to the left of the midclavicular line. Heart sounds were very faint; no murmurs audible; A2 > P2, but not accentuated. There was slight sclerosis of right temporal and radial arteries, not excessive considering the age of the patient. In general the heart sounds were of regular rhythm, although occasionally at the apex a pause approximately equal to the period of two beats was noted. No heart sounds were audible during this pause, which the patient recognized as an irregularity, because of the heart throb which followed. On the first examination of the patient in June, 1916, the pulse rate was 68; the blood-pressure: systolic 118, diastolic 68. On August 1, during the period of acceleration without palpitation or other symptoms, the rate varied between 105 and 120; blood-pressure: systolic 96, diastolic 65. November 22 the pulse rate averaged 95 between attacks, while in two different paroxysmal attacks it was 136 and 122 respectively. The lungs showed a few scattered medium moist rales. There was present moderate stethoscopic edema.

Urine. Acid: 1.015 to 1.020; albumin, 0 to very slight trace; sugar, 0; occasional hyaline cast; slight trace of pus. Phenolsulphonephthalein test 78 per cent. excreted in two hours.

The first electrocardiogram on this case was taken June 20, 1916, at which time the patient had no acceleration of rate (Fig. 1). It showed a normal origin and conduction of impulses with an average rate of 76 beats per minute. The *P–R* interval was 0.16 second; the *P* waves were upright in leads I and II, diphasic in lead III. There was a slight left ventricular preponderance.

August 1 several electrocardiograms were obtained. During the taking of one of these the patient was having an acceleration of rate without any symptoms, such as he said "occurs about once in a thousand times." This electrocardiogram (Fig. 2) showed a simple acceleration with a rate of 107 to the minute. The *P* waves were upright, as before, in leads I and II (the only leads taken), while the *P–R* interval was unchanged, 0.14 to 0.16 second, indicating a normal origin and conduction of impulses. Another plate, taken the same day, showed a rate of 76 with a *P–R* interval of 0.16 second and upright *P* waves.

November 22 the patient was again electrocardiographed during normal periods and during two typical attacks of acceleration, such

as have been described in the case history. During the normal periods the rate varied from 73 to 100, averaging about 95; the *P* waves were upright in all leads and preceded the ventricular deflection by 0.16 second.

During the attacks the rates were 136 and 122 respectively, the *P* wave was invert and following the *R* wave, with an *R–P* interval of 0.1 second. There was no change in the ventricular complex during the paroxysms, the onset and offset of which were abrupt.

FIG. 1.—(No. 210-1, lead II.) Electrocardiogram of Case I taken during a period of normal mechanism. The rate was 76. *P–R* interval = 0.16 second. The ordinates of all electrocardiograms shown represent 10^{-4} volt, the heavy and fine abscissæ represent time intervals of 0.2 and 0.04 second respectively.

With the return to normal in the last paroxysm the rate dropped suddenly from 122 to 73, the *P* wave became upright, at first low then gradually increasing in height, and there was an immediate return to a *P–R* interval of 0.16 second (Fig. 3). Between the paroxysmal attacks of tachycardia the regular rhythm was occasionally interrupted by a premature beat of junctional origin which showed the normal form of ventricular complex, except that it was

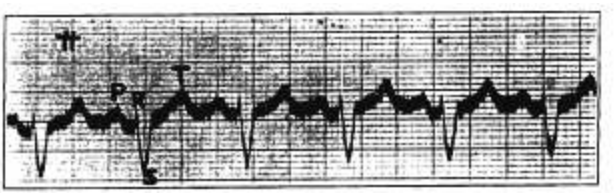

FIG. 2.—(No. 210-5, lead II.) Electrocardiogram of Case I taken during a period of normal mechanism with acceleration. No subjective symptoms. The rate was 107. *P–R* interval = 0.14 to 0.16 second.

of diminished size (Fig. 4). The *P* wave was invert and followed the *R* wave by 0.1 second. The pause following the premature beat was almost compensatory. During these premature beats no sounds were heard over the precordium and no pulse could be felt at the wrist.

The second case described cannot be definitely classified. There

are several points in favor of the impulse formation being of heterogenetic type, others in favor of a homogenetic origin. The nodal rhythm developed while the patient was under ether anesthesia,

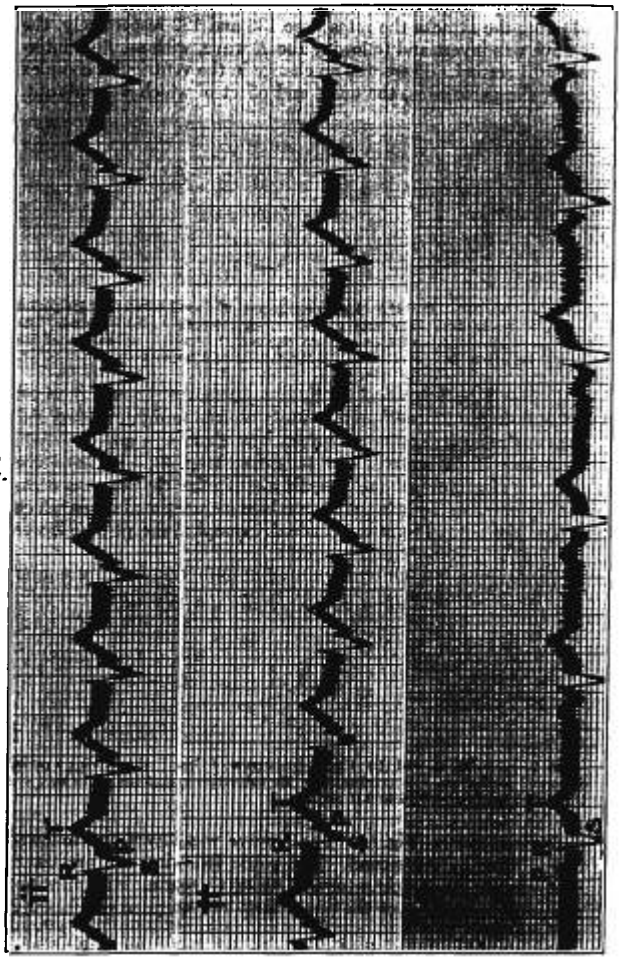

FIG. 3

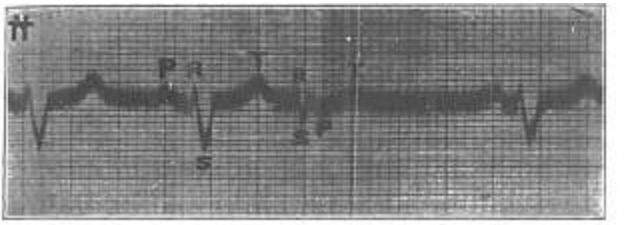

FIG 4.—(No. 210–10a, lead II, film.) Electrocardiogram taken a few minutes after Fig. 3. A junctional premature beat is shown in which the ventricular complex is smaller than but of the same general conformation as that of the normal cycles, the *P* wave being invert and following the *R*.

and neither the onset nor offset of the abnormal mechanism was observed. The clinical history of this case follows:

CASE II.—Mrs. M. W., aged thirty-six years, white; married; housewife. Admitted to St. Francis Hospital, service of Dr. H., January 2, discharged January 30, 1917.

Chief Complaint. About fourteen months before admission patient had noticed gradually increasing general weakness, which became so marked that any effort caused extreme fatigue; she had been confined to her home during that period, spending most of the time in a chair or in bed. The weakness had steadily increased despite rest in bed and medical treatment. For two and one half years preceding she had noticed occasional weakness, but it had not been so severe. About eight months before coming to the hospital the patient tried to increase effort and took two walks of about 300 yards each; both were followed by a "fainting attack" similar to others she had had for the past sixteen years. These attacks always began with a feeling of thoracic oppression, as though there was a weight on the chest; patient's lips and fingertips became very cyanotic; a cyanotic lacework appeared over the skin; the hands grew cold; and patient became very dyspneic and weak. Because of weakness she had accustomed herself to lying down; she had lost consciousness only two or three times, and that many years before. She never had had convulsions, but attacks

EXPLANATION OF FIG. 3

(No. 210–10, lead II.) Electrocardiogram of Case I taken during a typical attack of paroxysmal tachycardia. The entire figure is of lead II, the three parts being taken in immediate succession. The end of the paroxysm occurred while the camera plate was being raised for the taking of the third portion of the figure. The postparoxysmal pause was observed but not recorded. A period of about five seconds elapsed between the end of each third of the figure and the beginning of the succeeding third. The heart rate during the paroxysm was 122 with an *R–P* interval of 0.1 second. The rate after the paroxysm was 73 with a *P–R* interval of 0.16 second. Note the similarity of the ventricular complex during and after the attack, except as it is modified by the superimposition of the invert *P* during the paroxysm. There is a progressive increase in the height of *P* immediately following the paroxysm.

ended with a "nervous chill." During these attacks, because of the dyspnea and weakness, patient lay in a semicomatose condition, knowing what went on about her, but unable to speak. The extreme dyspnea usually lasted from fifteen minutes to an hour, during which time there was gradual improvement. The weakness lasted two or three days. During these attacks, according to statement of husband, the pulse was usually "better" than between them; by this statement he means stronger and faster; it was never below 44 nor over 60, but usually irregular. The description of this irregularity is much like that of a premature beat, followed by compensatory pause, while the description of the irregularity of pulse noticed between attacks is more like an actual "missed beat." Pulse has been slow during the patient's entire life, usually below 60. Last year, for one month, pulse is said to have been constantly between 18 and 25, and was noted on several occasions by her attending physician at these rates. She never has had palpitation nor edema. For years she has had slight unproductive cough, not increased with dyspnea. Her nervousness had been noticed for seven years; she became easily excited and had "internal quivering," followed by increased weakness and dyspnea. Frequently she had paresthesia of arms and legs, usually bilateral, but never any local paresis nor paralysis. The paresthesia usually lasted fifteen to twenty minutes, and was often, though not always, associated with dyspnea. Recently the feeling of "pins and needles" had been noticed whenever the patient awoke at night. There was always dysmenorrhea; menses were irregular. The "cardiac attacks" noted above usually come just before or just after the menstrual period. Nycturia once; diuria 6 to 7. No dysuria nor hematuria; chronic constipation; occasional gaseous eructation. For the past six months the patient had had an eruption on the face.

Past History. Measles, mumps, and varicella as child. At the age of two years the left eye was injured and the sight of this eye was destroyed. Frequent attacks of tonsillitis until ten years ago, when they ceased.

At sixteen years had the first "fainting attack," similar to that described in present illness. These attacks had occurred about twice a year since that time, but more often since present illness. There had been frequent partial aphonia since girlhood.

At nineteen years, she had a severe hemorrhage, but whether gastric or pulmonary was not known, and no details were obtainable.

In 1911 she had left-sided dry pleurisy and a "nervous breakdown," because of which she was in a hospital for fifteen weeks.

For twenty years has had dyspnea on slight exertion (one flight of stairs) even between attacks.

Occupational History. School teacher and supervisor for sixteen years until two years ago; since then has had household duties. She always worked very hard.

Family History. Practically negative. Married two years. Never pregnant.

Physical Examination. Slightly built, poorly-nourished woman lying flat in bed in no discomfort. Skin of left half of face showed many discrete and confluent dark red papules; only one papule on right side of face.

Mucous membranes of good color; not cyanotic.

Left eye showed internal squint; patient could only distinguish light with this eye.

Oral cavity and throat negative, save for crowded teeth and dental work.

Lungs were negative.

Heart outline was small to percussion. *R. B. D., 3 cm.; L. B. D.,* 7 cm. from midsternal line in IV interspace. Apex felt in fourth space, 7.5 cm. from midsternum. Supracardiac dulness 4.5 cm. wide. Heart sounds were of good quality, regular except for a very rare "missed beat," during which pause no sounds were audible over precordium. This pause was about twice the length of that between the regular beats. At pulmonic area a soft, blowing systolic murmur was audible; not transmitted. $P2 > A2$ not accentuated. No thrills. Pulse was of fair volume and low tension; the walls of the radials were not sclerosed. Rate, 56 to 63. Blood-pressure: systolic, 100; diastolic, 60.

Abdomen: Negative. Knee-jerks: Lively. Extremities: No edema.

Pelvic examination by Dr. Huggins revealed an infantile anteflexed uterus and undersized ovaries.

Laboratory Findings. Blood: Hemoglobin, 80 per cent.; red blood cells, 4,460,000; white blood cells, 7400. Morphology of reds and differential essentially normal.

Urine: Negative.

Phenolsulphonephthalein test: Sixty-seven per cent excreted in two hours.

Wassermann (blood): Positive.

Wassermann on husband's blood negative (never received antispecific treatment).

Course. Temperature usually subnormal.

Pulse varied from 40 to 126, usually between 40 and 70 with a tendency to remain at about 60. During most observations the pulse was regular except for the occasional presence of a long pause, during which no heart sounds were audible. The frequency of these pauses varied from day to day, sometimes recurring every few beats, at other times unrecognizable during a prolonged period of observation. When the pauses were most frequent the pulse was slowest. The slowest rate observed by writers was 41, although the husband is said to have observed a rate of 32 shortly previous to this time.

January 20, under ether, patient had dilatation and curettage of uterus, with introduction of stem pessary by Dr. Huggins. The

pulse and blood-pressure findings at this time will be given in Table I. Pessary later came out and was reinserted January 27 without ether. During stay in hospital patient complained of general weakness like that which had preceded admission. She had several periods of paresthesia, as previously described, but objective examination at such times revealed nothing definite.

The results of electrocardiographic examinations will be discussed in the body of the paper. Patient discharged January 30, only slightly improved.

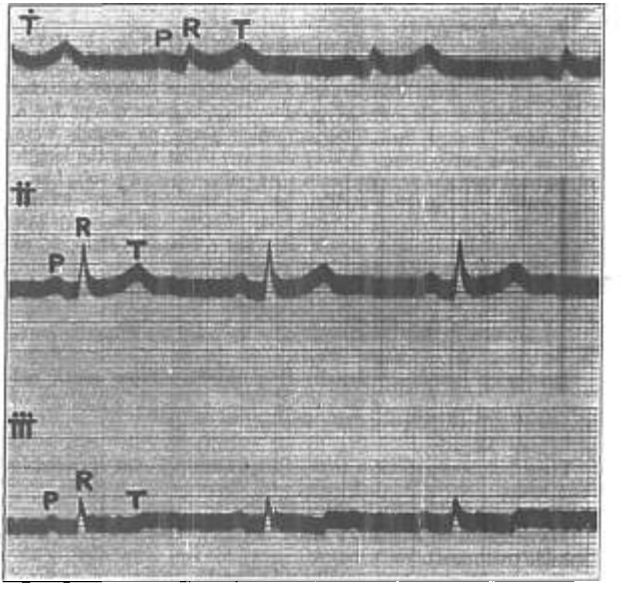

FIG. 5.—(No. 308–11, leads I, II, III.) Electrocardiogram of Case II taken during a period of regular heart action. There is a slight sinus arrhythmia. Rate = 55. *P–R* interval = 0.16 second.

On seven different occasions during the stay in the hospital, electrocardiographic examinations were made. On all these occasions, except while the patient was under ether, the electrocardiograms showed a marked sinus arrhythmia, frequent sino-auricular heart-block, and bradycardia (Figs. 5 and 6). The slowest rate obtained by electrocardiograph was 41, the longest *R–R* interval was 2.04 seconds. The *P–R* interval varied from 0.16 to 0.18 second during the normal rhythm. The *P* wave was

always upright in leads *I* and *II* and upright or diphasic in lead III. In the electrocardiogram taken immediately after the operation, January 20, while the patient was still under complete ether anesthesia, the mechanism was different from that at any other

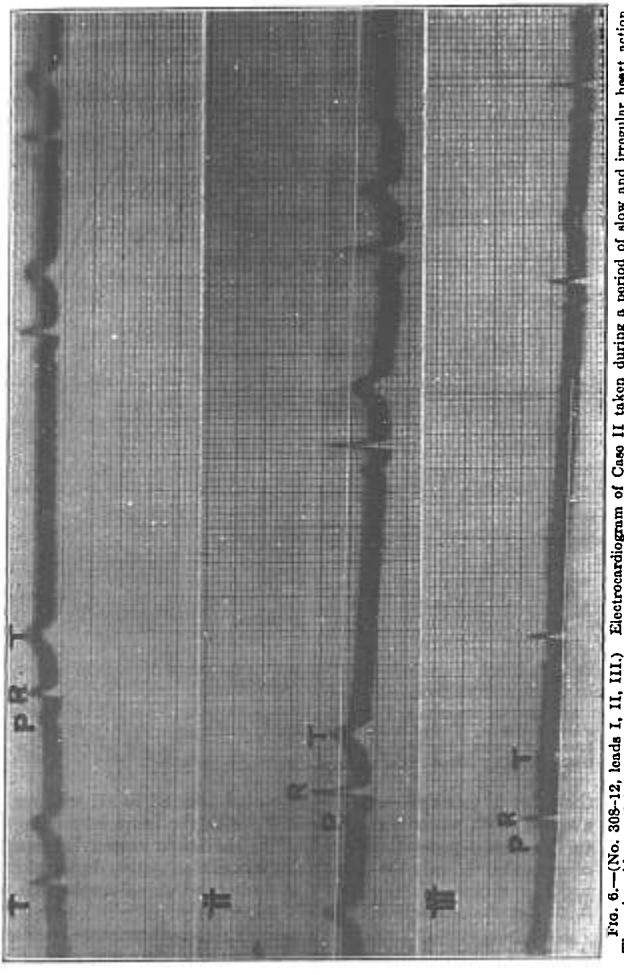

FIG. 6.—(No. 308–12, leads I, II, III.) Electrocardiogram of Case II taken during a period of slow and irregular heart action. There is evidence of frequent sino-auricular block. The average ratio is 41, the longest *R–R* interval = 2.04 seconds. The *P–R* interval = 0.16 to 0.18 second.

examination (Fig. 7). There was no marked change in the ventricular complexes except that the *R* waves were slightly higher, while the *T* of lead III tended more toward inversion. The *P* wave, however, instead of preceding the *R* followed it with an *R–P* interval of 0.06 to 0.1 second. The contour of the *P* was also changed, for in this plate it was of smaller amplitude and shorter duration, while in all leads it varied from upright to diphasic. The rate of

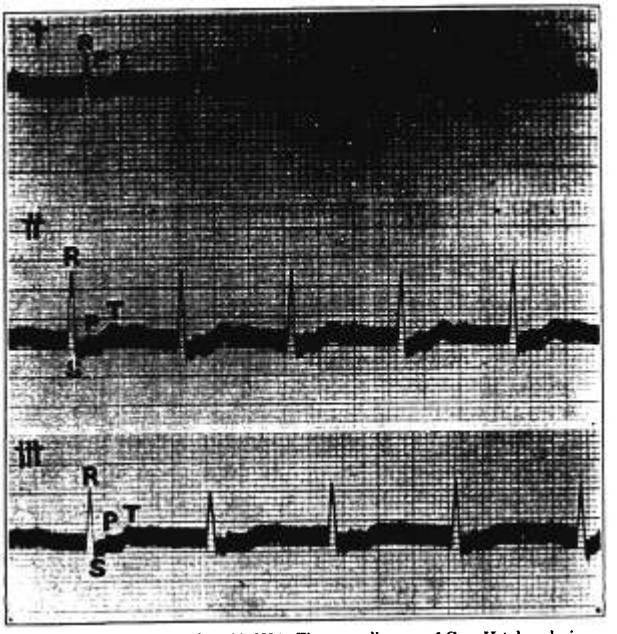

FIG. 7.—(No. 308–8, leads I, II, III.) Electrocardiogram of Case II taken during ether anesthesia. The pacemaker has shifted from the sino-auricular node to the junctional tissues. The heart rate varies from 97 to 83. The *R–P* interval varies from 0.06 to 0.1 second. The ventricular complex is unchanged.

the heart in this electrocardiogram varied from 83 to 97, with an average of 92, whereas the fastest rate with the heart responding to the normal pacemaker was 63. As one might expect because of the enhanced rate the duration of ventricular systole during the nodal rhythm was shorter than that during the normal mechanism, 0.28 to 0.32 and 0.4 to 0.44 second respectively.

Unfortunately the record taken during the operation was not developed until several hours later, so that the change in mechanism

was not known at once. For this reason the series is not so complete as it might be desired. As soon as it was known there had been a shifting of the pacemaker the patient was again electrocardiographed, but not until five hours after the preceding plate had been made. Meanwhile there had been complete recovery from the anesthetic. This latter group of plates showed a return to the normal mechanism, the *P* upright in all leads, *P–R* interval 0.14 to 0.16 second, the rate 63, with a slight sinus arrhythmia, but no evidence of sino-auricular block. All the deflections at this time were very small. The next electrocardiograms showed a return to the condition before the operation, a condition that persisted throughout all remaining electrocardiograms.

A table of the pulse and blood-pressure readings on the day of operation may be of interest (Table I). There was a marked variation in the intensity of the systolic sounds over the brachial artery while blood-pressures were being taken during the operation. The intensity varied with the phases of respiration, being much the louder during inspiration. This phasic variation was most marked during early anesthetization, but it persisted throughout the period of etherization and for some time thereafter.

We have already stated that the first case cited was of the heterogenetic type. The fact that the paroxysms could sometimes be stopped by the patient either by holding the breath or by rapid, deep breathing would seem to indicate that the control of the extrinsic cardiac nerves was not entirely lost. However, as these effects were inconstant, we must beware of the *post* and *ergo propter* fallacy. The other criteria for the recognition of junctional paroxysmal tachycardia, namely, rapid rate, sudden onset and offset, shortened, absent, or negative *P–R* interval, inversion of *P*, normal ventricular complex, and postparoxysmal pause were all present.

In the second case neither onset nor offset was pictured or observed. From the pulse rates as given in Table I, because of the length of the intervals between observations, we cannot be certain whether or not the change was sudden. However, inasmuch as the rate during the taking of the electrocardiogram varied from 97 to 83, while there was a general tendency toward a gradual fall both before and after the electrocardiogram was taken, the supposition is that the change was not abrupt. The rate, although much more rapid than during response to the sinus pacemaker, was not excessively rapid when the electrocardiogram was being taken. However, it may be presumed that the nodal rhythm was also present at an earlier stage of the operation when the rate was 120. From the data at hand we cannot determine whether the rhythm in this second case was of heterogenetic or homogenetic origin. However, as the latter origin is much the more common we favor the supposition that the disturbance was of the homogenetic type in spite of the fact that the comparatively rapid rate is in favor of the former.

TABLE I.

Jan. 20. 1917. Time.	Systolic.	Diastolic.	Pulse-pressure.	Pulse rate.	Character of pulse.	Notes.
9.00 A.M.	104	66	38	63	Regular	Before operation.
9.30	..	..	..	..	..	Morph. sulph. gr. $\frac{1}{4}$; atrop. sulph. gr. $\frac{1}{60}$.
10.30	..	..	..	80	Regular	Ether begun.
10.50	112	95	17	120	"	Partial anesthesia.
10.53	140	110	30	110	"	Partial anesthesia.
10.55	125	110	15	114	"	Complete anesthesia
11.00	132	70	62	126	"	Dilatation of cervix begun.
11.05	130	65	65	123	"	Curettage.
11.08	..	..	..	95	"	Operation finished.
11.10	..	..	..	..	..	Electrocardiogram taken; complete anesthesia.
11.15	135	95	40	..	..	Patient removed from table.
11.30	..	..	..	90	"	Patient in her room.
11.40	..	..	..	80	"	
12.00	..	..	..	64	"	Patient partially recovered from ether.
12.05 P.M.	..	..	..	70	"	
12.30	..	..	..	80	"	
12.40	..	..	..	68	"	
12.50	..	..	..	70	"	
1.05	110	70	40	60	"	Still drowsy from ether.
1.15	..	..	..	68	"	
1.25	..	..	..	70	"	
1.55	..	..	..	68	"	
2.20	..	..	..	68	"	
4.15	100	65	35	68	"	Completely recovered from ether; electrocardiogram taken.
6.00	..	..	..	..	..	Morph. bimeconate, gr. $\frac{1}{4}$ s.c.
8.00	..	..	..	60	"	Feeling comfortable.

The question of etiology is an interesting one—several factors require consideration. Thus the patient had been given a hypodermic of morphin (gr. $\frac{1}{6}$) and atropin (gr. $\frac{1}{150}$) one hour before etherization was begun. We know of no observations in regard to morphin administration in connection with observed instances of shifting of the pacemaker to the conducting tissues. The case is different with regard to atropin. Thus, Wilson was able to induce nodal rhythm by vagus stimulation in normal persons who had received preliminary injections of atropin (1 mg.). This result could usually be obtained only when stimulation was applied eight to twenty minutes after the injection. In our case the electrocardiogram was taken one hour and forty minutes after the injection, and but twenty minutes after the marked rise in rate noted in Table I. This suggests that the shifting of pacemaker was not

the result of the morphin-atropin injection alone. Furthermore, the administration of the morphin-atropin mixture is a routine measure preliminary to operations in St. Francis Hospital, and no other cases of nodal rhythm have been observed by us over a series of 21 cases in which electrocardiograms have been taken during anesthesia. That asphyxia is capable of causing a shifting of pacemaker to *A–V* node has been shown by Lewis, White, and Meakins[8] who induced nodal rhythm in cats by this method. However, since our patient took the ether well and at no time showed any signs of asphyxiation we must look further for the cause of the changed mechanism. Wilson[9] cites 3 cases in which forced respiration caused the pacemaker to change from the *S–A* to the *A–V* node. As the respirations were increased under ether administration this forcing may have been a partial factor in the changed mechanism. However, the data is too scanty to permit of definite conclusions being drawn. The effect of the ether itself is not fully known, but our other cases show that, at least, it is not commonly a cause of *A–V* rhythm.

In most cases of nodal rhythm the *P* is invert; but its inversion is not an essential characteristic. Thus Meakins[10] has shown that the *P* wave may be polyphasic, and that in only 8 of his 9 experiments did the auricular complex begin with a downstroke. Lewis and Allen[11] have described a case of premature beats arising in the *A–V* tissues in which case the *P* wave was upright and of normal contour; from this fact, together with the varying *P–R* intervals, they conclude that the auricles were responding to the normal pacemaker while the ventricles were responding to an impulse arising low in the *A–V* bundle. This case, however, is not analogous to ours. In our first case the *P* wave was invert; it was upright or diphasic in our second. We believe that both are true cases of nodal rhythm, the auricles and ventricles both responding to the new pacemaker.

Wilson divides each of his previously mentioned types of nodal rhythm into three subtypes dependent upon the origin of the impulse: (1) those with a shortened *P–R* interval, *P–R* 0.1 second or less; (2) those in which the *P–R* interval is absent, *P–R* equals 0; (3) those in which the *P–R* is negative, that is, changed to an *R–P* interval. The origin is supposed to be progressively lower in the auriculoventricular tissue in types 1 to 3; the latter is the lowest and also clinically and experimentally the least common. White[12] believes that an *R–P* interval may be due either to a low

[8] The Susceptible Region in *A–V* Conduction, Heart, 1914, v. No. 3, 289–298.

[9] Three Cases showing Changes in the Localisation of the Cardiac Pacemaker Associated with Respiration, Arch. Int. Med., 1915, xvi, No. 1, 86–97.

[10] Experimental Heart-block, with Atrioventricular Rhythm, Heart, 1914, v, No. 3, 281–288.

[11] An Instance of Premature Beats arising in the Auriculoventricular Bundle of a Young Child, Am. Jour. Med. Sc., 1913, cxlv, No. 5, 667–671.

[12] A Study of *A–V* Rhythm following Auricular Flutter, Arch. Int. Med., 1915, xvi, No. 4, 517–535.

position in the *A–V* tissues or to resistance in the auriculonodal junction or to both together. Hering,[13] however, has shown that the greatest delay in conduction through the junctional tissues occurs in the node of Tawara. The general belief is that with an *R–P* interval the origin of impulse is in or below the node. In both the cases we have described in this paper there was an *R–P* interval indicating a low origin in the junctional tissues; likewise, in both cases the ventricular complexes were unchanged, except for modification by the superimposition of the *P* wave, indicating that the impulses originated above the division of the main stem of the His bundle.

The slow rate of the sino-auricular rhythm in the second case is interesting in conjunction with the case of junctional paroxysmal tachycardia described by Lewis, in which the rate between attacks was usually about 50, with an occasional rate as low as 37. In regard to that case Lewis said that the slow rate occurred during a "sinus arrhythmia." Although the published curves do not show a sino-auricular block, we are led to question whether such a block were not occasionally present as in our case.

Eyster and Meek[14] have shown that the average rate of rhythmic discharge of the auriculoventricular tissues in dogs is about 67 per cent. of that of the sino-auricular node; in cats the relationship is closer but the rate is still below that of the *S–A* node; in man the automatic rhythmicity of the nodal tissues is likewise lower than that of the normal pacemaker. Hence, White suggests that when the rate of a nodal rhythm is rapid, that rapidity indicates more than a mere depression of the sino-auricular node; it indicates stimulation of the auriculoventricular tissue. The fact that the rates during nodal rhythm in both of our cases were high compared with the rates during the normal mechanism points therefore to some abnormal stimulation of the new pacemaker.

SUMMARY. Two cases are presented in which electrocardiographic study showed the development of transient nodal rhythm. The first case exhibited junctional premature contractions and typical attacks of paroxysmal tachycardia such as had extended over a period of twenty years; the second presented marked sino-auricular block, and under ether anesthesia there developed a paroxysm of auriculoventricular rhythm.

During the paroxysms of abnormal rhythm there was present in both cases an *R–P* interval indicating an origin of impulses low in the conduction tissues. The comparatively rapid rates during the paroxysms of auriculoventricular rhythm suggest an abnormal stimulation of the junctional tissues.

[13] Nachweis, dass die Vergögerung der Erregungsüberleitung zwischen Vorhof und Kammern des Säugerthierherzens im Tawaraschen Knoten erfolgt, Arch. f. d. ges. Physiol., 1910, cxxxi, 379–401.

[14] Experiments on the Origin and Conduction of the Cardiac Impulse. VII Sinoventricular and Sino-auricular Heart-block, Arch. Int. Med., 1917, xix, No. 1, 117–139.